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**14. ABSTRACT**
Electrical muscle stimulation (EMS) is used to strengthen muscles in rehabilitation of patients and training of athletes. Torque generation from evoked muscle contractions can equal torque from maximal voluntary muscle contractions. Voluntary muscle straining or an inflated anti-G suit increase the arterial blood pressure (Bp) and give a pilot G-protection during increased +Gz. This work was designed to determine if arterial blood pressure increases during normal gravity conditions with lower body electrical muscle stimulation. The intention was also to develop an efficient and comfortable elastic suit with new mesh electrodes sewn into the suit.

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Electrical muscle simulation (EMS), pilot G-protection, voluntary muscle straining, anti G-straining manuever

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SHORT COMMUNICATION

# An Electrical Muscle Stimulation Suit for Increasing Blood Pressure

ULF BALLDIN, LANCE ANNICELLI, JOHN GIBBONS, AND JAMES KISNER

BALLDIN U, ANNICELLI L, GIBBONS J, KISNER J. *An electrical muscle stimulation suit for increasing blood pressure.* **Aviat Space Environ Med 2008; 79:914–8.**

***Background:*** Electrical muscle stimulation (EMS) is used to strengthen muscles in rehabilitation of patients and for training of athletes. Voluntary muscle straining and an inflated anti-G suit increase the arterial blood pressure (BP) and give a pilot G protection during increased $+G_z$. This study's aim was to measure whether BP also increases with EMS of lower body muscles. ***Methods:*** A suit with new cloth electrodes sewn into the garment was developed. There were 12 subjects who were tested in sitting position during 3 conditions with 10 consecutive periods of EMS, inflated anti-G suit (GS), or lower body muscle anti-G straining maneuvers (AGSM). BP was continuously measured noninvasively. ***Results:*** The means of the baseline systolic BP, before each of the test conditions, were 127 ± 16, 128 ± 11, and 145 ± 14 mmHg for GS, AGSM, and EMS, respectively. During inflation of the GS, execution of the AGSM, and EMS, mean systolic BP during the first 10 s was 143 ± 15, 146 ± 13, and 150 ± 13 mmHg, respectively, with no statistical difference between the conditions. The corresponding mean resting heart rate before each test was 57–63 bpm for all conditions. During the test periods with GS, AGSM, and EMS, heart rate was 59 ± 11, 79 ± 16, and 61 ± 15 bpm, respectively, with statistical differences ($P < 0.001$) between AGSM and the other two conditions. ***Conclusion:*** EMS created similar BP as GS and AGSM at 1 G and also had higher pre- and post-control values. Further studies are required to evaluate if this principle may be used for G protection of pilots.

**Keywords:** electrical muscle stimulation, muscle straining, G-suit, blood pressure.

ELECTRICAL MUSCLE stimulation (EMS) is used to strengthen muscles in rehabilitation of patients and training of athletes. Torque generation from evoked muscle contractions can equal torque from maximal voluntary muscle contractions (13). Voluntary muscle straining or an inflated anti-G suit increase the arterial blood pressure (BP) and give a pilot G protection during increased $+G_z$ (5). This work studied the arterial blood pressure effects during normal gravity conditions with lower body EMS using an elastic suit with new mesh electrodes sewn into the suit.

The current anti-G straining maneuver (AGSM) consists of both isometric muscle contractions of the lower body and abdominal muscles as well as a cyclic breathing maneuver against a closed glottis (Valsalva maneuver) with increased intrathoracic pressure (5). The voluntary contractions of the peripheral muscles, which induce peripheral vasoconstriction of the arteries and arterioles, together with the respiratory straining maneuver, will increase the heart level blood pressure. An AGSM can provide G protection by increasing the blood pressure at heart and cerebral level (5). This reduces the risk of gray-out, blackout, and G-induced loss of consciousness during high G loads. However, this technique is extremely fatiguing and requires the pilot to anticipate the change in acceleration and forcefully engage his skeletal muscles (2). The AGSM is accompanied by the use of an anti-G suit (GS), which acts by compressing the lower extremities and abdomen to reduce blood pooling to the lower body. This compression of the lower body muscles induces a peripheral vasoconstriction, resulting in increased blood pressure (5), which adds G protection (8). The GS's bladders are currently inflated through an anti-G valve supplied by compressed gas. It can take up to 3–4 s to fully inflate the bladders of the GS, a long time when exposed to rapid-onset, high G loads. The bladders are rather bulky when inflated and can cause thermal stress.

In the quest for new principles of G protection, a literature search showed that electrical stimulation of muscles has successfully been used to improve muscle strength in rehabilitation after surgery (e.g., knee surgery), in rehabilitation after spinal cord injury and strokes, and in muscle strength training of athletes (10–12). In a review article by Gentakow (7), electrical stimulation was described as improving ischemic wound healing and circulatory response in both animal and human studies. However, there is very little information about the effects of electrical muscle stimulation on systemic arterial blood pressure. The literature search did not indicate that anyone has tried electrical stimulation of muscles as a means of increasing systemic arterial blood pressure with the goal of improving G tolerance during exposure to high G loads. Therefore, in a first

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step, this study was intended to find out if there is an effect on blood pressure from electrical muscle stimulation using a newly developed suit with elastic mesh electrodes sewn to the inside of the suit. A further goal was to compare these effects to those seen from execution of lower body muscle straining maneuvers (as in a partial anti-G straining maneuver used by fighter pilots) and from G-suit inflation.

Preliminary exploratory information from our laboratory indicated that certain frequencies of electrical stimulation by skin electrodes on the lower body muscles and abdominal muscles may increase systolic arterial blood pressure. The preliminary results indicated a potential for continuing this study. The hypothesis was that electrical muscle stimulation of the lower body muscles induced with a muscle stimulation suit increases systemic arterial blood pressure to a similar level as muscular straining maneuvers of the lower body muscles and as an inflated G suit without exposure to increased acceleration.

## METHODS

Before starting the study, different frequencies were initially tested using electrical muscle stimulation on the investigators. Original large electrodes attached to the proximal and distal parts of the calves and thighs and over the abdominal muscles with elastic bands were used for these registrations. A commercially available electrical muscle stimulator (Tone-A-Matic, Inc., Buffalo, NY) used for muscle strength training in rehabilitation and by athletes was used. Frequencies from 10 to 39 Hz (called Tens), and from 50, 60, 70, 75, 100 to 200 Hz, and the so-called Russian stimulation of 2500 Hz were tested in an exploratory way in about 100 trials. Maximal individual stimulation intensity was selected to give a solid, tetanic muscle contraction without being painful. The arterial blood pressure increases from baseline were registered with noninvasive Portapres® equipment (FMS, Amsterdam, The Netherlands) with a probe attached to a finger of the hand and with reference level to the heart. With frequencies of 70 and 75 Hz, systolic blood pressure increases up to 60 mmHg and diastolic up to 30 mmHg were registered. From these exploratory measurements a frequency of 70 Hz was selected as a potentially effective method to increase arterial blood pressure.

An undergarment suit consisting of pants and a T-shirt (Body Conditioning Gear, BCG®, Academy, Katy, TX) made of 80–87% nylon and 13–20% spandex was used for the electrical stimulation condition. New elastic knitted Electro-Mesh® highly conductive electrodes using Intelligent Textiles for Medicine® with silver treated nylon fibers blended with Dacron (Prizm Medical, Inc., Duluth, Georgia) were sewn into the garment. The elastic mesh electrodes were circumferentially placed proximally and distally on the calves and thighs and over the gluteal and abdominal muscles to create a positive and negative pole over the muscle areas. For better electrical contact between the electrodes and the skin, the electrodes were wetted with water before the experiment.

For this study, the electrical biphase stimulation with 70 Hz frequency was then individually tested to find the optimal, but not uncomfortable, stimulation intensity level in each subject. The maximum stimulation for the electrical muscle stimulation with 9-V (DC) battery-operated stimulation did not exceed 300 V (AC) with low amperage distributed over a large surface area in bursts of about 140 μs for every 1 s of stimulation. This stimulation intensity corresponds to what is commonly used in muscle straining and muscle rehabilitation programs (13).

The risk of electrical muscle stimulation was minimal in the way it was done in this study. The electrical stimulators were battery-operated and the stimulations were of sufficiently low amperage to avoid any apparent harmful effects at the levels we used. The subjects felt the stimulation as a vibrating sensation and felt the contractions of the muscle. If the muscles were not ordinarily trained or only slightly used, there could be some minor sensation of soreness after the stimulation, similar to muscle strain after voluntary contractions of muscles not used very much or not used with high intensity. Such stimulations are currently used in rehabilitation therapy and in muscle strength training in athletes. However, if the electrical stimulation is too intense, the result will be muscle contraction pain. Each subject was instructed to have the investigator lower the intensity or stop the stimulation if muscle contraction pain was experienced.

There were 12 volunteer (1 female and 11 male) members of the Laboratory's centrifuge subject panel who participated in the study. Their mean age was 31 yr (range 20–42), mean height 177 cm (range 140–193) and mean weight 81 kg (range 61–109). The protocol was approved by the Brooks Institutional Review Board and all subjects signed an informed consent document. Each subject's activity, food, and fluid intake the day prior to each test was ad libitum, except for alcohol, which was prohibited.

Each subject participated in three experimental sessions: GS inflation to 13.8 kPa (2 psi), lower body muscle AGSM, and EMS. The sessions occurred on different days, and the order of the exposures was randomized and balanced. At each session, arterial blood pressure (systolic and diastolic) and heart rate were measured for 30 s before, 30 s during, and 30 s after the test condition with the Portapres (FMS) noninvasive continuous arterial blood pressure measuring technique on a finger of the right hand with reference level to the heart. The subjects were sitting upright on a mock-up ejection seat with no footrest during the tests. This sequence was performed a total of 10 times, with a 2-min break between each sequence. The averages of 20 s ending 5 s before the completion of the precontrol period, the first 10 s (5–15 s) and the last 20 s (5-25 s) during the 30-s treatment period, and 20 s of the post-control period (starting after 5 s in the post-control period) were kept for data analysis. Subjects were asked to be quiet and breathe normally during the measurements, since talking increases thoracic pressure somewhat, which in turn increases the

arterial blood pressure. The total time for each session took about 30 min to complete for each subject. For the AGSM session, since muscle straining for G protection usually also involves Valsalva maneuvers with raised intrathoracic pressure (which would increase BP), we required that the muscle strain be performed with an open glottis to avoid intrathoracic pressure increase. To ascertain that the glottis was open the subjects were connected to a pneumo-tachograph to verify normal breathing during the straining maneuvers. The subjects were trained centrifuge subjects used to executing the straining maneuver during high G exposures in the centrifuge. They were instructed to do a normal muscular straining maneuver with their leg and abdominal muscles (without a Valsalva maneuver).

In addition to the physiologic measures, subjective fatigue scores were also obtained from each subject after each of the 10 trials of each experimental session. The subjective fatigue survey used an 11 point scale with 0 = nothing at all, 0.5 = very, very low (just noticeable), 1 = very low, 2 = low (light), ...., and 11 = maximal.

Initially, exploratory tests were performed using analysis of variance to determine whether there were any trends over the 10 sequential trials at each session that should be taken into consideration for further analysis. No such trends were detected and we, therefore, averaged over the 10 trials for each subject under each condition. These data became the "raw" data for statistical analysis. A repeated measures analysis of variance (ANOVA) with two within-subject factors (condition and time) was performed on each outcome measure separately. If significant condition by time interactions were detected, post hoc comparisons using Student's paired *t*-tests were used to test for changes from pre-control at each time point, and to compare all pair-wise combinations of the three experimental conditions at each data collection time point. Since this was an exploratory investigation, we chose not to use conservative "multiple comparison" adjustments when performing the tests. All testing was performed at the 0.05 alpha level.

## RESULTS

For each of the three outcome measures (systolic BP, diastolic BP, and heart rate), significant condition by time interaction was detected by the ANOVA. Thus, results of the post hoc comparisons will be presented for each measure below.

Systolic blood pressure (**Fig. 1**) started at 145 ± 14 mmHg under the EMS condition and remained essentially flat during the first 10 s (150 ± 13 mmHg) and last 20 s (148 ± 12 mmHg) and after (146 ± 13 mmHg) the exposure. GS and AGSM systolic pressures started lower than EMS pressures (127 ± 16 and 128 ± 11 mmHg, respectively), and significantly climbed to approximately the EMS levels during the first 10 s (143 ± 15 mmHg and 146 ± 13, respectively) and last 20 s (138 ± 15 mmHg and 143 ± 13) of the exposure. GS pressure returned to pre-control levels after exposure (128 ± 18 mmHg).

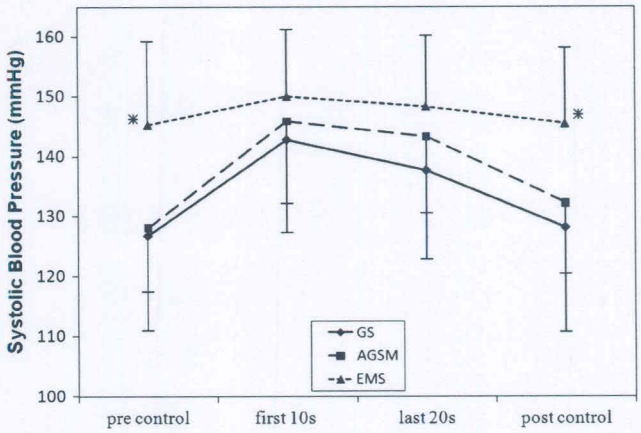


**Fig. 1.** Mean systolic blood pressures (± SD) in the pre-control period during the first 10 s and last 20 s of treatment, and in the post-control period during G-suit inflation (GS), lower body muscle straining (AGSM), and electrical muscle stimulation (EMS). The * indicates that the blood pressure values during EMS were statistically different from the GS and AGSM ($P < 0.05$).

AGSM pressure also dropped after exposure, but remained slightly, but significantly, higher (132 ± 12 mmHg) than pre-control pressure. Comparisons at each time point indicated that EMS systolic pressure was significantly higher than the GS and AGSM pressures at pre-control and post-control, but no differences were found during exposure.

Diastolic pressure (**Fig. 2**) for EMS started at 78 ± 9 mmHg, remained fairly flat during the first 10 s (81 ± 10 mmHg) and last 20 s (79 ± 10 mmHg) of exposure, and fell slightly, but significantly (to 76 ± 9 mmHg), below that of pre-control after exposure. GS and AGSM diastolic pressures started lower (70 ± 12 mmHg and 69 ± 10, respectively), climbed significantly to levels comparable to EMS levels during the first 10 s (83 ± 13 mmHg and 80 ± 10, respectively) and the last 20 s (78 ± 12

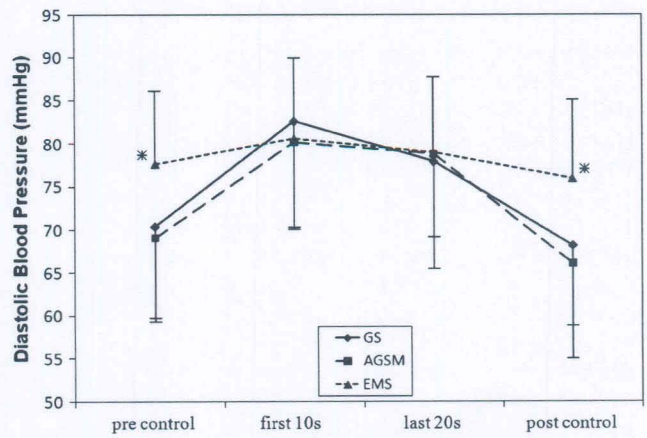


**Fig. 2.** Mean diastolic blood pressures (± SD) in the pre-control period, during the first 10 s and last 20 s of treatment, and in the post-control period during G-suit inflation (GS), lower body muscle straining (AGSM), and electrical muscle stimulation (EMS). The * indicates that the blood pressure values during EMS were statistically different from the AGSM ($P < 0.05$).

mmHg and 79 ± 10, respectively), and fell to levels that were slightly, but significantly, lower than pre-control levels after exposure (68 ± 13 mmHg and 66 ± 9, respectively). Comparisons at each time point found that AGSM pressure was significantly lower than EMS pressure before and after exposure. GS pressure exhibited the same pattern, but the differences did not reach statistical significance.

EMS heart rate started at 57 ± 12 bpm, increased slightly but significantly during the first 10 s (62 ± 16 bpm) and last 20 s (61 ± 15 bpm) during the exposure, and was 59 ± 11 bpm after exposure. GS heart rate started at 59 ± 12 bpm, remained flat at the first 10 s (58 ± 10 bpm) and last 20 s (59 ± 11 bpm) of exposure, and showed a slight but significant increase (to 64 ± 13 bpm) after exposure. AGSM heart rate, on the other hand, started slightly higher (63 ± 12 bpm), showed a large significant increase during the first 10 s (to 75 ± 14 bpm) and last 20 s (79 ± 16 bpm), and remained significantly elevated after exposure (at 74 ± 15 bpm). When comparing the conditions at each time, AGSM heart rate was significantly higher than EMS heart rate at all four test points, and was significantly higher than GS heart rate at all but the pre-control point. Finally, the GS heart rate was significantly higher than the EMS heart rate at the post-control test point.

The mean subjective ratings of fatigue with use of the above-mentioned 11-unit scale were overall very low and showed only a slightly higher mean value for the muscle straining maneuvers, where it was just under 1.3 units (very low). All the other responses were under 0.5 unit (very, very low).

## DISCUSSION

Sparse information from the literature indicates that the arterial systolic blood pressure may be increased in animal models and in humans using electrical muscle stimulation. In a study by Hultman and Sjöholm (6) of blood pressure and heart rate response to voluntary and non-voluntary static exercise in man, a systolic blood pressure increase by 30% and diastolic increase by 50% was reported during percutaneous electrical stimulation of the thighs. Davies and Starkie (4) found that systolic and diastolic blood pressure increased linearly throughout muscle contractions, and systolic blood pressure increased more rapidly than the diastolic. There was no significant difference in response between electrically stimulated and voluntary muscle contractions. Electrical stimulation of muscles has been used after G-exposures to test if anti-G straining maneuvers are mainly restricted by central or peripheral fatigue (1). High-voltage pulsed galvanic stimulation was shown by Heath and Gibbs (9) to increase calf muscle blood flow in humans.

Theoretically, for a physically well-conditioned person, a voluntary muscle contraction eliciting a systemic arterial blood pressure increase, as when executing a muscle straining maneuver for G protection or mechanical muscle compression induced by a GS, should have the same effect as electrically induced (non-voluntary) muscle contraction. The muscle contraction and the muscle compression induce a mechanical pressure to the underlying arteries and arterioles, causing a reduction in blood vessel diameter. That will increase the peripheral resistance, and with an unchanged cardiac output, an increase in the systemic arterial blood pressure will be the result. Added to this there is a slowly developing, reflex-induced increase in blood pressure (5). This end result of the changes in systemic blood pressure was also the finding in this study. Voluntary muscle contraction (as with the straining maneuver) and muscle compression (as induced by an inflated GS) caused a higher blood pressure than in a resting control state. Electrical muscle stimulation created systemic arterial blood pressures that were similar to those produced by a GS and a lower body muscle AGSM at 1 G, but electrical muscle stimulation also caused higher pressures during pre- and post-controls.

There is a direct relationship between intrathoracic pressure and systemic arterial blood pressure (3). The pneumo-tachographic control of open airways (no Valsalva maneuver and no talking) that was employed during the muscle-straining maneuver assured that an increased intrathoracic pressure did not influence and further augment the reported blood pressure increases. The inflation of the GS was kept at a relatively low level (13.8 kPa or 2 psi), which is the standard pressure used for pressure tests of a GS before exposure in the human centrifuge. This, however, is much lower than the pressure that can exist at such high G levels as +9 $G_z$, where it can reach 70 kPa. But such a high pressure is not necessary to induce a blood pressure increase at lower G levels and can be quite painful at 1 G.

The higher baseline blood pressure before the electrical muscle stimulation may have been due to subject apprehension or a residual effect from the earlier electrical stimulation during the calibration phase and during the preceding stimulation in the 10 consecutive stimulations with 3 min between the stimulations (30-s post-control measurement, 2-min rest, followed by 30-s pre-control measurement). The higher control value after each stimulation followed the same pattern, indicating a longer-lasting effect of the electrical muscle stimulation than just for the stimulation phase. Blood pressure did not seem to be lower after each stimulation period, indicating that the apprehension factor probably was not as pronounced as the longer-lasting electrical stimulation effect. This residual effect of the electrical stimulation may explain why blood pressure did not increase as much as with muscle straining and GS inflation. However, the end result was that blood pressure was as high with electrical stimulation as with straining or GS inflation.

With the new electrical muscle stimulation suit, at the beginning of the study, we found that the contact between the electrodes and the skin was at times marginal, resulting in a reduced effect from the stimulation and/or a tingling or sticking sensation in the skin. This problem was avoided by wetting the electrodes with water before use.

The developed electrical muscle stimulation equipment may have a potential for G protection of pilots

through its effect of increasing blood pressure. We are planning a separate study to verify this. This prototype suit, compared to present G suits, has the benefits of being less bulky and heavy, causing less thermal burden than a standard anti-G suit, and alleviating the need for a gas pressure supply. Other possible applications may be to counteract blood pressure fall in patients with orthostatic hypotension or after return from long spaceflights with cardiovascular de-conditioning.

Electrical muscle stimulation produced systemic arterial blood pressure levels that were at least equal to those generated by GS inflation and AGSM with lower body muscles at 1 G. Further studies are required to evaluate if electrical muscle stimulation also may have a potential for G protection of pilots, for protection against hypotension after long-lasting space travel, or to protect patients with postural hypotension.

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REFERENCES

1. Bain B, Jacobs I, Buick F. Is there central fatigue during simulated air combat maneuvering? Aviat Space Environ Med 1995; 66:1–5.
2. Balldin UI, Werchan PM, French J, Self B. Endurance and performance during multiple intense high +Gz exposures with efficient anti-G protection. Aviat Space Environ Med 2003; 74:303–8.
3. Balldin UI, Wranne B. Hemodynamic effects of extreme positive pressure breathing using a two-pressure flying suit. Aviat Space Environ Med 1980; 51:851–5.
4. Davies CT, Starkie DW. The pressor response to voluntary and electrically evoked isometric contractions in man. Eur J Appl Physiol Occup Physiol 1985; 53:359–63.
5. Green NDC. Protection against long duration acceleration. In: Ernsting J, Nicholson AN, Rainford DJ, eds. Aviation medicine, third ed. Oxford, England: Butterworth and Heineman; 1999:148–56.
6. Hultman E, Sjoholm H. Blood pressure and heart rate response to voluntary and nonvoluntary static exercise in man. Acta Physiol Scand 1982; 115:499–501.
7. Gentzkow GD. Review: Electrical stimulation for dermal wound healing. Wounds: A compendium of clinical research and practice 1992; 4(6):227–35.
8. Glaister DH, Prior RJ. The effects of long duration acceleration. In: Ernsting J, Nicholson AN, Rainford DJ, eds. Aviation medicine, third ed. Oxford, England: Butterworth and Heineman; 1999:128–47.
9. Heath ME, Gibbs SB. High-voltage pulsed galvanic stimulation: effect of frequency of current on blood flow in the human calf muscle. Clin Sci 1992; 82:607–13.
10. Milner-Brown HS, Miller RG. Muscle strengthening through electric stimulation combined with low-resistance weights in patients with neuromuscular disorders. Arch Phys Med Rehabil 1988; 69:20–4.
11. Ratkevicius A, Skurvydas A, Povilonis E, Quistorff B, Lexell J. Effects of contraction duration on low-frequency fatigue in voluntary and electrical induced exercise of quadriceps muscle in humans. Eur J Appl Physiol Occup Physiol 1998; 77:462–8.
12. Russ DW, Vanderborne K, Binder-Macleod SA. Factors in fatigue during intermittent electrical stimulation of human skeletal muscle. J Appl Physiol 2002; 93:469–78.
13. Wong RA. High voltage versus low voltage electrical stimulation. Force of induced muscle contraction and perceived discomfort in healthy subjects. Phys Ther 1986; 66:1209–14.